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THE HEART AND SUDDEN DEATH.

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THE HEART

AND

SUDDEN DEATH

BY

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PREFACE.

THIS little book comprises in the main some articles which appeared in *The Hospital.* They are based upon experience gained in performing nearly 2,500 autopsies. Although the morbid anatomy of the heart in its relation to sudden death is chiefly dealt with, it is hoped that some of the observations here contained may not only be of interest to medical men when considering disease of the heart in its broader aspects, but may be of aid to medical students in calling to mind facts not always clearly dealt with in text-books of medicine.

T. F.

May 1, 1908.

CONTENTS.

THE HEART AND SUDDEN DEATH.

THE MITRAL VALVE.

WHEN we speak of sudden death, both medical men and those without medical training generally think at once of the heart. And it is needless to say that they are right to regard it as all-important to the maintenance of life. The presence or absence of the beat of the heart for more than a few moments of time is synonymous with the absence or presence of death. In passing, it may be remarked that formerly, in reports of inquests in the daily papers—and occasionally I think it is so still—apoplexy is mentioned as a cause of sudden death. Absolutely sudden death from such a cause, however, is extremely rare. It is necessary to remember that cardiac action is virtually independent of the brain. The emotions of the mind acting through the brain may disturb the heart's rhythm, and such impulses may temporarily, or in very rare instances even permanently, arrest cardiac action. Yet the brain itself is not responsible for the steady continuance of cardiac systole and diastole, as it is for the respiratory movement of the chest; and consequently if an

acute lesion of the brain is to produce sudden death, it must either cause it by shock to the heart or by injuring the respiratory centre. Should respiration cease, cardiac action, it is needless to say, will cease soon also from want of aëration of the blood which flows through the coronary arteries, but hæmorrhage into the brain rarely occasions death quickly in this way.

Fallacies Regarding the Mitral Valve.

The most common causes of arrest of cardiac action arise not without the heart, but within it. In connection with these causes arising within the heart, however, there are possible fallacies. Perhaps the most common is the importance attached to what is called fatty degeneration of the heart. Again, although the words "mitral regurgitation" are not often used in coroners' courts, this morbid condition is another stumbling-block. Mitral regurgitation, it is true, is not considered to be a common cause of sudden death, yet the importance of regurgitation through the mitral orifice, as a disease, is deeply ingrained in the minds of most of us; and this mental attitude affects the way those of little experience examine a heart in which they expect to find the cause of death. There being no fatty degeneration of the cardiac muscle that they can detect, and no disease of the aortic valve, they examine the cusps of the mitral valve, and almost invariably succeed in discovering something abnormal which they consider sufficient to account for death. The most common

error will be referred to later, but it may not be out of place briefly to refer to the size of the mitral orifice. Some students appear to be taught that the mitral orifice normally admits the tips of two fingers. It is scarcely necessary to remark that the tips of fingers differ much in size; yet when we consider how the height and weight of the human body varies, the size of the normal mitral orifice is remarkably constant. It ranges only very slightly above and below 4 inches in circumference. The average size of two finger-tips is a little over 3 inches, whereas three finger-tips measure about 4 inches. Three finger-tips are frequently stated—and it seems to me are more correctly stated than two—to be a gauge of the size of a normal mitral orifice.

Probably, however, few of those inexperienced in post-mortem work lay stress upon evidence of dilatation of the mitral orifice as measured by the finger-tips. There is a much more common source of error. This error is the discovery of a thickened edge to the mitral valve. The expression "edge of mitral valve thickened" is a familiar one, and the reason why such thickening is so often thought to be present and to be the consequence of disease is because one feature of the anatomy of the mitral valve is not sufficiently borne in mind. The cusps of the mitral valve are composed of layers of endocardium covering strands of fibrous tissue, and the point to which I wish to draw attention is that these fibrous strands are prolongations of the chordæ tendineæ. The chordæ tendineæ, on becoming inserted into the

flaps of the mitral valve, become minutely subdivided, and their fibres interlace in their course towards the mitral ring at the base of the flaps. The interlacing of these fibres occurs chiefly just above the margin of the valve, and in the situation of this interlacing there is a band of thickening. During the process of examination of the heart this thickening is often made to appear greater than it really is. When incising the heart, in order to obtain a good view of the flaps of the mitral valve, not only is the left ventricle laid open, but the mitral orifice is divided between the flaps, without, however, severing the connections of the chordæ tendineæ with the muscular pillars. Whether the heart is then held in the hand or is placed on the post-mortem table, abnormal tension is placed on some of the chordæ tendineæ, and this tension causes puckering of the edge of the large flap of the mitral valve. Let this flap, however, be held up against the light, that is, so that the light is seen through it, and all the apparent thickening and abnormal puckering will resolve themselves into the normal structure of the valve. The course of the divided fibres of the chordæ tendineæ will then be clearly seen, and the part the fibres play in producing the thickening which is normally present be made evident.

The Nature of Inflammation of the Mitral Valve.

Having indicated a mistake that is to be avoided, we may next consider in what way inflammation

affects the mitral valve. The early evidences of inflammation, it is needless to remark, are small vegetations over the position of contact of the edges of the flaps. These are, however, in cases of rheumatic endocarditis of no importance in themselves. If inflammation were limited to the site of the vegetations, endocarditis would not be followed by serious consequences. Unfortunately the inflammation extends far more widely in too many instances. It generally affects the fibrous structures of the chordæ tendineæ from their origin at the apices of the musculi papillares to their insertion into the ring at the base of the flaps of the mitral valve. This inflammation is also often progressive, that is to say, it does not end with the attack of rheumatic fever in the course of which it originated. The continual movement of the valve probably acts as a source of irritation to the formation of new fibrous tissue. This newly formed fibrous tissue eventually contracts, but does not produce puckering of the mitral valve. Puckering is a sequel of irregular inflammation. The inflammation which succeeds rheumatic endocarditis is not irregular; it spreads more or less uniformly over the whole of the mitral flaps and along the chordæ tendineæ connected with them. Inflammation of more irregular character, it may be mentioned, not uncommonly occurs in association with infective endocarditis. Infective endocarditis is not by any means always acute; it is sometimes of long duration, and may heal in one place while it spreads in another. As a consequence of such local healing,

very definite puckering may be produced. But to return to rheumatic endocarditis, the newly formed fibrous tissue in the chordæ tendineæ and in the valve contracts, and contracts uniformly, with the resulting effect that the edges of the flaps are drawn

FIG. 1.—EARLY RHEUMATIC DISEASE OF THE MITRAL VALVE SHOWING THE CHORDÆ TENDINEÆ FREE FROM THICKENING.

The letter F is placed just above the line of attachment of the lesser flap of the mitral valve, and points by a dotted line to the larger flap. The letter C is placed on chordæ tendineæ, passing to the lesser flap, and the letter T is situated on the chordæ tendineæ attached to half of the margin of the larger flap.

downwards towards the apex of the ventricle and inwards towards one another.

The difference in length and thickness of the chordæ tendineæ in the early and late stages of rheumatic endocarditis will be made evident by a

reference to the accompanying photographs. Fig. 1 shows rheumatic vegetations, which, it may be remarked in passing, are unusually large for vegetations of rheumatic origin; but the chordæ tendineæ are little, if in any degree, thickened. Fig. 2 shows the

FIG. 2.—RHEUMATIC DISEASE OF THE MITRAL VALVE OF LONG STANDING, SHOWING GREAT SHORTENING OF THE CHORDÆ TENDINEÆ.

The letter F is placed on the large flap of the mitral valve. Immediately below most of the chordæ tendineæ have entirely disappeared.

large flap of the mitral valve drawn down until it is in contact with one of the muscular pillars, and the contraction of fibrous tissue which has produced this has caused the disappearance of many of the chordæ tendineæ. These features of inflammation of the mitral valve are apparently not widely recognised,

and it seems that teachers of medicine often speak as if unaware of the important part played by the chordæ tendineæ in disease of the mitral valve. It is not too much to say that *rheumatic disease of the mitral valve is mainly a disease of the chordæ tendineæ.*[1] It has previously been mentioned that the contraction of the chordæ tendineæ draws the flaps of the mitral valve downwards and inwards towards one another—in other words, it produces a narrowing of the orifice, or what we know as mitral stenosis. The flaps are, however, generally prevented from closing efficiently, and regurgitation through the orifice is also therefore present. During life the regurgitant murmur alone may attract attention, where after death the most important condition found proves to be mitral stenosis. We may in this connection make another statement, and say that when in a case of sudden death *there is no evidence of stenosis of the mitral orifice, any abnormality of the mitral orifice that may be found has probably had little or nothing to do with the cause of death.* It is necessary, however, to add that uncomplicated mitral stenosis is a very rare cause of sudden death.

Before leaving the subject of mitral stenosis it should be mentioned that Dr. Ewart has described a case of what he calls "soft-valve mitral stenosis" in which the valve and chordæ tendineæ were not thickened. I have not met with such a case. It

[1] Although most teachers of medicine apparently speak of the importance of mitral regurgitation following puckering of the flaps of the mitral valve, Dr. Graham Steell has long expressed the opinion that this view is not borne out by cases seen in the post-mortem room.

seems possible that the condition may have been of congenital origin. In a case of congenital mitral stenosis, however, which came under my notice the appearances of the valve were scarcely similar to those described by Dr. Ewart.

Sudden Death in Mitral Regurgitation.

When examining a case of sudden cardiac failure after death a preconceived idea that the mitral valve has probably been at fault may lead to the real seat of the disease in the heart being overlooked. On one occasion when I was making incisions into the cardiac muscle a highly qualified young medical man, who had worked in a pathological laboratory under a distinguished bacteriologist, remarked that in his experience of post-mortem work he had never seen the heart-wall carefully examined, and had never seen fibroid disease of the heart until he had witnessed me demonstrate its presence. Allowing that his statement was correct and his own powers of observation had not been wanting in accuracy, there is little doubt that he must have seen many cases of death wrongly attributed to mitral regurgitation.

The mitral valve, it is needless to remark, frequently becomes incompetent where the cardiac muscle presents some deviation from the normal, but the slight leakage which occurs through the valve must play a very small part in derangement of the circulation and the risk it occasions of sudden arrest of the circulation cannot but be very small indeed. Disease of the cardiac muscle usually occupies a very different position. When such disease is present the varied functions

of the muscle, although they may apparently be working smoothly, are always on the verge of various degrees of disturbance, and possibly without warning may be abolished for ever. In the majority of such cases the disease of the muscle-wall of the heart is evident after death to the naked eye. Yet the heart-wall may appear healthy, as there will be occasion to state later.

It may be mentioned in this connection that rheumatism, although it mainly attacks the valves, possibly not very uncommonly interferes with the nutrition of the cardiac muscle without producing abnormal appearances that can be recognised by the naked eye or even sometimes by the microscope. Occasionally, such naked-eye or microscopical lesions are very evident (see Fig. 10), but there are other cases in which it appears that the cardiac muscle must have been at fault, where no very definite evidence of disease can be detected. Some people who have had rheumatism, although no murmurs indicative of valvular disease are audible, are liable to attacks of dyspnœa, which may be accompanied by pain.[1] In the subjects of these attacks a sudden fright or a slighter cause, such as some annoyance, may start the dyspnœa. This association of dyspnœic attacks with an exciting cause acting upon the "nerves" may lead

[1] I have known attacks of pain and dyspnœa following rheumatism to occur not only in young women but in a boy aged nine where there was no evidence of valvular disease. The pain was apparently severe, and the boy used to call out for someone to press upon the chest over the region of the heart during the attacks.

the friends or medical attendant to consider the outbursts to be nothing but hysteria. Yet, while I do not think that attacks of dyspnœa following rheumatism are necessarily due to hysteria, at the same time, it seems to me to be doubtful at least when there is no evidence of valvular disease, whether they are usually of serious import; but in order to express a very definite opinion upon such a point, cases would need to have been watched for many years. It may be of interest to add here that it seems possible that a nervous shock may sometimes seriously weaken the heart for a considerable time where there has been no history of previous rheumatism. Several cases of this nature have come under my notice.

In one instance a girl, aged twenty-one, was introduced by a lodging-house keeper, either through some strange misunderstanding or from astonishing and callous thoughtlessness, into a bedroom where the young man to whom she was engaged was lying dead. The girl knew he was ill, but apparently, when she entered the room, was not even aware that he had been in any great danger. The sudden shock of learning the truth in this cruel manner produced so much disturbance of the heart that she was unable to leave the house for two hours, at the end of which time, by the aid of a friend, she was taken home. The following day, dyspnœa and cardiac pain returned. When she came under my notice, several months later, she was still subject to these attacks, and on walking fast or going upstairs she suffered from breathlessness. I have met with similar cases following such incidents

as the presence of burglars in a house, the jumping of a large dog on to the patient's back, and after seeing a relative fall down in an epileptic fit. In one of these cases swelling of the legs was associated with the symptoms of cardiac weakness. This seems to show that, in this case at least, there was something more than a neurosis. It is easy to consider any morbid condition that appears obscure to be purely functional, and although it may be tempting to take this view of weakness of the heart following fright, it seems to me to be possible that unusually strong impulses, such as must occur in exceptional conditions of nervous shock, may affect the nutrition of the heart, just as some progressive lesions of the central nervous system appear occasionally to originate in a peripheral injury. It is interesting to note in this connection that there are recorded cases of death some days after a severe shock has been received. A pathetic instance is the death of Vanessa, who is said to have died a week after Swift had angrily, in person, returned the letter she had written to Stella.

We are, however, wandering somewhat from the question of sudden death associated with lesions of the mitral valve. It has been previously mentioned that sudden death associated with definite disease of the valve is rare, and that where such death occurs it does not necessarily follow that the disease of the valve has been mainly responsible for the fatal issue. We have only lightly touched upon the importance of the cardiac muscle, but it may be mentioned here that fibroid patches, possibly as large as the little

finger-nail, are sometimes found in cases of death from mitral stenosis. Cases of death by slow cardiac failure are now referred to; but it is in cases in which such patches are present that sudden death would be most likely to occur. I do not happen to have had to investigate a case of sudden death from mitral stenosis, so that my own personal experience can only indicate probabilities. Something further will need to be said about the mitral orifice when sudden death associated with affections of the cardiac muscle is considered; but in closing these remarks upon disease of the cusps of the valve, it may be interesting to give an illustration of the apparently small inconvenience a serious lesion of the mitral valve may occasion even to a strenuous life. In a man, aged sixty-five, who had died of bronchitis, I found a mitral valve greatly thickened, and so stenosed that it only admitted the tip of one finger. On inquiry it was ascertained that the man had suffered from five attacks of rheumatic fever during the course of his life, yet apparently was unconscious of anything having been wrong with his heart. He had lived an active life as a horse trainer until within a few weeks of his death. It is needless to remark, however, that instances of this character are not frequently met with. The majority of those affected with mitral stenosis die between the ages of thirty and forty years.

THE AORTIC VALVE.

THE relation of diseases of the aortic valve to sudden cardiac failure is of a much more intimate character than is the case with disease of the mitral valve. There are, however, it is scarcely necessary to remark, varieties of diseases of the aortic valve, and the danger of sudden death is not equally great in all. It is well known that aortic regurgitation is a more serious disease than aortic stenosis, but possibly it is not always realised that there are varieties of aortic regurgitation, and that the risk to life is not the same in every variety. Dr. Seymour Taylor has thought that in cases where there is also mitral regurgitation the outlook is less serious than when disease is limited to the aortic valve, owing to the fact that dangerous over-distension of the left ventricle is relieved by escape of blood through the mitral orifice. That may be so, but there are other reasons why aortic regurgitation varies in the risk to life its presence entails.

THREE VARIETIES OF DISEASE OF THE VALVE.

Before indicating these reasons it may be interesting briefly to refer to various forms of disease of the valve. In the first place we have the results of

rheumatic endocarditis ; secondly, there is *thickening and deformity associated with disease of the aorta ;* and, thirdly, *a calcification of the segments of the valve* occurs which apparently is quite a distinct disease from atheroma of the aorta, and is most commonly present where there is no disease of this vessel.

Here it may be mentioned, however, that calcification is not confined to one form of endocarditis. Lime salts may be deposited in any chronic inflammation of the endocardium. In old-standing rheumatic disease, whether of the aortic or mitral valve, the presence of calcareous masses is not uncommon, yet there is a disease of the aortic valve in which early calcification is the most prominent feature. This disease sometimes leads to the formation of very large masses of calcareous material, which, it is needless to say, greatly interfere with the outflow of the blood from the ventricle. And even when these masses are not large, the segments of the valve may, by the deposit of salts, be rendered so rigid that they will only allow a very small stream to pass between them.

Calcification of the segments of the aortic valve is the most frequent cause of obstruction of the aortic orifice. It has been said that aortic stenosis unassociated with aortic regurgitation is extremely rare. The experience of most observers no doubt varies considerably, but it has happened that in the course of my post-mortem work pure aortic stenosis has not been very rare—at least I have met with nine cases in which there was no evidence of regurgitation.

Fig. 3 is a photograph from an example which occurred in a man aged fifty-five, who had cut his throat on account of urgent dyspnœa. It illustrates, what curiously enough appears to be not uncommon in these cases of calcareous disease, that the aortic valve is composed of only two segments. The lia-

FIG. 3.—CALCAREOUS DISEASE OF THE AORTIC VALVE.

The valve is viewed from above. There are only two valve-segments. The letter A, placed near the cut margin of the aorta, points by dotted lines to calcareous masses in these segments.

bility of the aortic valve when composed of two, instead of three, segments, to suffer from calcareous disease has attracted the attention of Dr. Parkes Weber and other observers. An interesting feature of this disease, also worthy of notice, is that, *although it is often spoken of as atheroma, the aorta is almost in-*

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large flap of the mitral valve drawn down until it is in contact with one of the muscular pillars, and the contraction of fibrous tissue which has produced this has caused the disappearance of many of the chordæ tendineæ. These features of inflammation of the mitral valve are apparently not widely recognised,

clearly seen in Fig. 4, which is given because it illustrates another feature of calcareous disease of the aortic valve. A calcareous process is seen extending downwards into the large flap of the mitral valve. Instead of extending into the mitral valve a similar process, or processes, may invade the wall of the heart; there is, in fact, a small process invading the septum between the ventricles in this case. Local extension suggests that the disease is of a locally infective character, and if locally infective it is strange that the aorta almost invariably remains healthy. This seems to be especially noteworthy because another variety of aortic valvular disease frequently appears to owe its existence to neighbouring disease in the aorta.

The most common cause of aortic stenosis is calcareous disease, but in rheumatic endocarditis also the segments of the valve may occasionally become adherent and thus produce stenosis though the valve remains competent. This, however, it is needless to say, is rare, because the associated fibroid thickening of the segments almost invariably causes them to shrink, and thus renders them incapable of meeting at the central point of contact. I could, however, give two illustrations of aortic stenosis, uncomplicated by regurgitation, due to disease of the valve following rheumatism.

Aortic regurgitation due to shrinkage of the valve-segments following rheumatism scarcely requires comment, but it may be of interest to give brief attention to another and important variety of re-

seems possible that the condition may have been of congenital origin. In a case of congenital mitral stenosis, however, which came under my notice the appearances of the valve were scarcely similar to those described by Dr. Ewart.

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valvular disease associated with disease of the aorta is most commonly met with in middle life. The calcareous variety of disease of the valve, in which, as

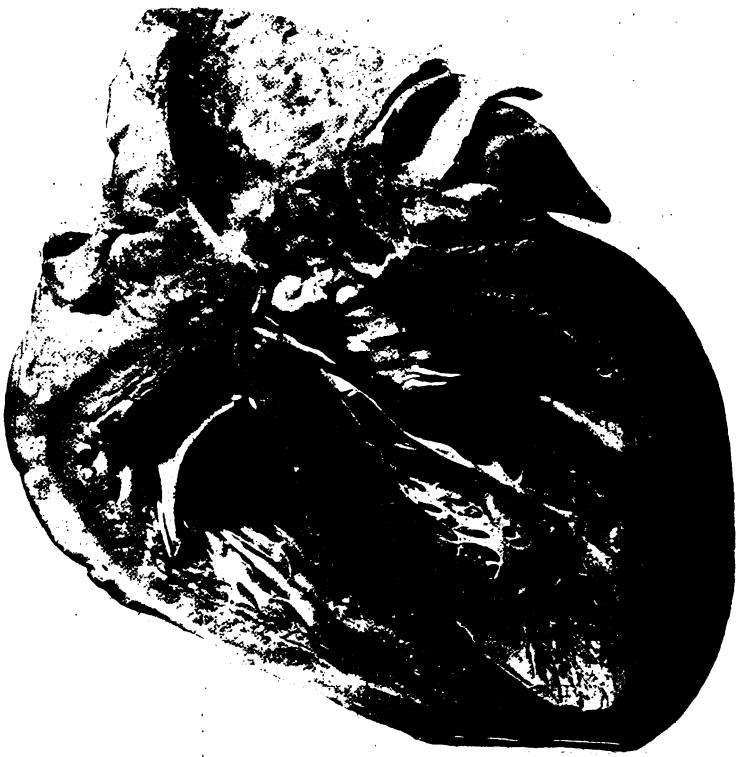

FIG. 5.—FIBROID DISEASE OF THE AORTIC VALVE ASSOCIATED WITH DISEASE OF THE AORTA.

has been previously mentioned, the aorta is healthy, occurs chiefly at a later age, but I have seen it in an adult under the age of thirty.

Regurgitation through an incompetent aortic valve, as is well known, frequently results in sudden death, but it is in cases where the aorta is also diseased that the danger is greatest. Not only does the presence of disease of the aorta greatly add to the danger of aortic regurgitation, but sudden death very commonly occurs where there is disease of the vessel-wall limited to an area which extends upwards for one or two inches above the aortic valve while the valve itself remains healthy and competent. A word of explanation may be necessary. Sudden death in aortic valvular disease has been frequently attributed solely to mechanical causes consequent upon leakage of the valve. Under conditions of extra strain the over-filled left ventricle has been thought permanently to cease to act in the midst of diastole, being unable to discharge contents which are unusually excessive in amount. Such may be the explanation in some cases, but most commonly it is not over-filling of the ventricle, but the condition of the cardiac muscle which is responsible for the abrupt arrest of cardiac action. The reason is as follows. Disease of the aorta situated immediately above the aortic valve frequently interferes with the circulation through the heart-wall by obstructing the orifices of the coronary arteries, and thus causes degeneration of the cardiac muscle. The weakened muscle may fail suddenly, and after death its cut surface will be seen to be studded with patches of fibrous tissue of various sizes, while microscopical examination generally shows also the presence of fatty changes. A point of interest

and importance is, therefore, that *the serious nature of aortic valvular disease associated with disease of the aorta is largely due to the fact that the disease of the aorta obstructs the orifices of the coronary arteries. As a consequence of this obstruction, the nutrition of the cardiac muscle suffers, fatty and fibroid changes occur which are a more serious menace to life than any variety of valvular disease.*

Disease of the cardiac muscle found in association with aortic valvular disease does not, however, occur only when disease of the aorta is present. It was long ago suggested that disease of the aortic valve may interfere with the circulation through the coronary arteries. At least one of these ideas has been shown to be erroneous; yet my experience would lead me to believe that the cardiac muscle presents serious morbid changes much more often in aortic valvular disease—whether the form be mainly that of stenosis or of regurgitation—than in diseases of the mitral valve. I refer now to cases of aortic valvular disease where the aorta is healthy. As an example, the case of a young man, aged eighteen, who fell dead while standing by the side of a horse may be mentioned. Thickening and adhesion of the aortic valve-segments were present and on cutting into the heart muscle extensive fibrosis was everywhere to be seen (see Fig. 6). At the age of eighteen any other cause for chronic valvular disease than rheumatism is rare, and the fibrosis of the cardiac muscle may have been a sequence of rheumatic myocarditis. Yet I have seen similar fibrosis in the heart of a middle-

aged virgin, where aortic stenosis of the calcareous type was present. I use the word " virgin " because the condition it describes virtually excluded the possibility of syphilis having been the explanation of

FIG. 6.—FIBROID DISEASE OF THE HEART-WALL ASSOCIATED WITH DISEASE OF THE AORTIC VALVE PROBABLY RHEUMATIC IN ORIGIN.

the condition of the cardiac wall, and the ordinary causes for fibrosis of the cardiac muscle were also absent. It seemed probable, therefore, that the heart-wall must have suffered in consequence of defective

nutrition dependent upon stenosis of the aortic valve. But whatever the cause of degeneration of the cardiac muscle in aortic valvular disease where the aorta is healthy may be, it is this degeneration of the muscle which is most to be feared when the question of sudden death is being considered.

Although such an accident as rupture of an aortic valve-segment has not, as far as I am aware, been recorded as a cause of sudden death, it may rapidly lead to a fatal issue, and a few words upon the condition may not be out of place. It has been thought that such an accident may occur in a perfectly healthy valve. This seems to me to be improbable. In most of the examples seen in museums, and also in the majority of recorded cases, there has been disease of the aorta. The disease of the aorta appears to have been of the character already referred to as the grey variety which is frequently associated with aortic regurgitation. Usually, as has been mentioned, the disease of the valve-segments that accompanies disease of the aorta is a fibroid thickening which leads to their shrinkage. Instead, however, of becoming thickened and shrunken, a valve-segment may bulge and rupture, or may become torn away from some portion of its attachment to the aorta. If we allow the disease of the aortic valve to be of the same nature as that of the aorta, the occasional presence of yielding and rupture is not surprising when we consider how commonly the disease of the aorta occasions aneurism. Fig. 7 is from a rough sketch of a specimen showing rupture of an aortic

valve-segment. Above the valve is seen puckering and thickening of the aorta due to disease. The specimen is from a man, aged forty, who was seized suddenly with dyspnœa ; but death did not occur until four months later. Another accident, somewhat similar to rupture and occurring also in associa-

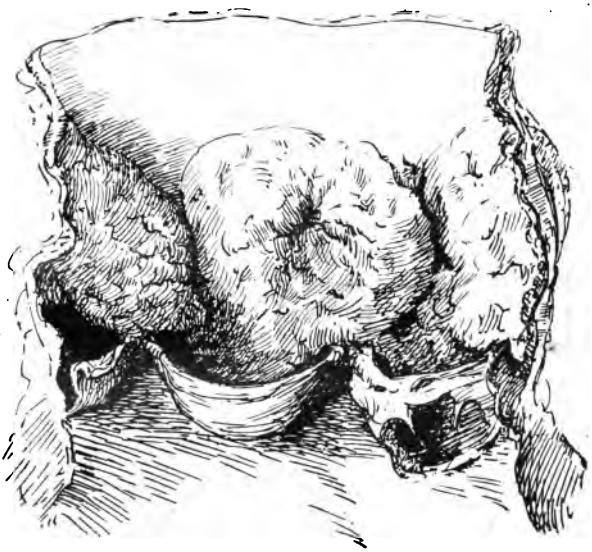

FIG. 7.—A ROUGH SKETCH SHEWING RUPTURE OF AN AORTIC VALVE SEGMENT ASSOCIATED WITH DISEASE OF THE AORTA.

tion with disease of the aorta, is retroversion of an aortic valve-segment. In one case which came under my notice the retroversion occurred in a travelling acrobat, who obtained his living by turning somersaults in the air and performing similar feats which require considerable effort. In his case the retro-

version of an aortic valve-segment, found after death, proved to be associated with disease of the first part of the aorta.

In closing these remarks upon diseases of the aortic valve and their relation to sudden death, a few points may be recapitulated.

Sudden death may, as is well known, occur in any variety of aortic regurgitation, and less frequently in aortic stenosis.

The form of aortic regurgitation in which sudden death is most common is that in which there is also disease of the first part of the ascending aorta, and the abrupt arrest of cardiac action is mainly due to disease of the heart-wall.

In other cases of aortic valvular disease in which sudden death occurs, whether the variety be of the calcareous type, or whether it be that which follows rheumatism, fibrosis of the cardiac muscle will probably be present.

THE CARDIAC MUSCLE.

In reports of inquests a frequent cause for death is said to be "fatty heart". There is generally much misunderstanding in the minds of those who use the term as to the nature of a fatty heart. A heart in which the main morbid condition present is what has been called fatty degeneration, is comparatively rare, and except as a complication of diphtheria or as an association of fibroid disease of the heart, may be said to be very rarely if ever seen in cases of sudden death. In passing, a word as to the nature of fatty degeneration may not be out of place. Modern physiology seems to indicate that fat is not formed from proteids, and that consequently fat does not arise in cardiac muscle fibres as a result of destruction of the important elements of these fibres. Most of the fat which may become evident in certain conditions of disease may have been present in the heart before the disease arose, but changes occurring as the result of deleterious influences have rendered the fat visible to the naked eye or to histological methods of examination, or it may be that under these influences additional fat has been stored in the cardiac muscle. Whether, however, the presence of fat in the cardiac muscle is, or is not, an evidence of true degeneration should the deleterious influences

leading to its appearance act only for a short time and be removed before death takes place the heart generally completely recovers. In other words, fatty degeneration when existing alone—apart from any other forms of degeneration of the cardiac muscle —must be looked upon as a temporary abnormal condition; that is to say, *there is no such disease as fatty disease of the heart, which may last for months and years and be a source of danger to life.* Here a word needs to be said about "fatty infiltration" of the heart. Some text-books seem to indicate, and the records of published cases of death make it clear, that excess of fat covering the heart and existing in the interstitial structures within the heart-wall is often considered to be an evidence of cardiac weakness. There is, I believe, no evidence in favour of such a view. In very stout people the fat covering and infiltrating the heart may be excessive in amount, but any dyspnœa which may have been present on exertion during life must obviously have been due to the work entailed in moving an unusually heavy body. Of one fact it is at least necessary to have a clear understanding, that fatty infiltration is no indication of the existence of fatty degeneration. The fat lying between the muscle-fibres may occasionally be very great in amount, yet the muscle-fibres prove on microscopical examination to be quite healthy.

The "Flabby" Heart.

Another term which needs a word of comment is that of "flabby". While the designation "fatty"

indicates a pathological condition of the cardiac muscle that under certain conditions may exist, the term "flabby" has no pathological meaning. The heart may be found after death to be firm or flabby, but those who consider a soft condition of the heart to be an evidence of disease overlook the consequences of changes which occur in the body after death. The cardiac muscle, like the voluntary muscles, undergoes rigor mortis. During rigor mortis the muscle-wall of the ventricles contracts. The wall of the strong left ventricle in contracting generally expels most of the blood it contained at the time of death, but the weaker right ventricle more often fails to drive out so much of the blood within it. Consequently there may not infrequently be seen a firmly contracted and conical left ventricle, and a right ventricle more or less distended. Should rigor mortis have passed off instead of the firm heart with a conical left ventricle, there will be a soft heart, which, after removal from the body, flattens out when laid on the post-mortem table. *A "flabby" heart, therefore, is a heart from which rigor mortis has disappeared.* The early onset of flabbiness will depend not on the condition of the cardiac muscle, but upon the heat of the weather and the nature of the illness which has led to death. In enteric fever, for example, the heart is far more likely to be found to be flabby at the time of the autopsy than in cerebral hæmorrhage, not because there is fatty degeneration in the case of fever—any such degeneration, if present, will be trifling in amount—but because rigor mortis has already passed

away. It may be well to add that the presence or absence of rigor mortis in the heart must not be judged by the rigidity of the voluntary muscles. Rigor mortis may have passed away from the heart while it is still present in the voluntary muscles.

To return to fatty degeneration. It is scarcely

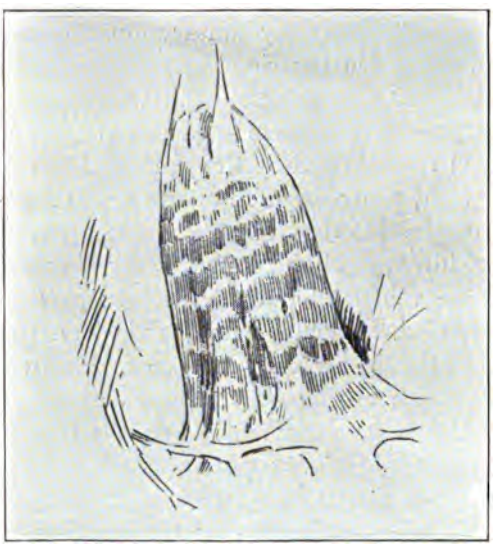

FIG. 8.—"TABBY-CAT" STRIATION OF A MUSCULAR PILLAR.

necessary to refer to the well-known appearance known as "tabby-cat striation" (see Fig. 8), which is most commonly seen in death following anæmia due to severe loss of blood, especially when a septicæmia is associated with the anæmia, such as may occur in puerperal fever following post-partum

hæmorrhage. "Tabby-cat" striation is also not uncommonly seen in cases of rheumatic pericarditis when the pericarditis has existed for two or three weeks or longer, but diseases such as rheumatism which occasion poisoning of the cardiac muscle more frequently give rise to a diffuse fatty degeneration.

FIG. 9.—FATTY CARDIAC MUSCLE-FIBRES IN RHEUMATISM.

The microscopical section is a thick one, the heart-muscle having been cut fresh, but the comparatively uniform distribution of the fat droplets through the muscle-fibres can be seen.

Pale, ill-defined patches are then sometimes seen in the heart-wall, the nature of which does not become clear until examination by histological methods has been undertaken, though in many cases nothing abnormal can be detected with the naked eye where

microscopical examination shows fat to be present. Fatty degeneration of this character occurs in diphtheria, yet in diphtheria where there has been sudden death the amount of fat present in the cardiac muscle is not always very noteworthy, probably because death has occurred before sufficient time has elapsed for the pathological changes to become evident. In some cases of sudden death in diphtheria death is, however, hastened by paralysis of the diaphragm, and in others possibly, as Bolton thinks, poisoning of nerve centres in the floor of the fourth ventricle may play a part in the fatal issue. The accompanying micro-photograph (see Fig. 9) has been inserted to illustrate the nature of fatty changes which occur in the cardiac muscle in such cases as rheumatism and diphtheria. This photograph is of a microscopical section of the heart-wall of a girl, aged fifteen, who died after three months' illness in which swelling of the joints and rheumatic nodules were present. The presence of a few adhesions over the auricles showed that there had been pericarditis of limited character, but there was no trace of inflammation of the pericardium over the ventricles. Numerous microscopical sections were made from various parts of the heart-wall, but no muscle-fibre could be discovered which did not contain minute globules of fat throughout its entire length. In "tabby-cat" striation the appearance is different. There microscopical examination shows groups of muscle-fibres almost replaced by fat while the majority of the fibres are quite healthy. In chorea similar disease of the

cardiac muscle may be seen to that which may occur in rheumatism. In the pathological museum of the annual meeting of the British Medical Association, held at Swansea in 1903, I showed microscopical sections of the heart-wall from a fatal case of chorea in which pericarditis had not been present. Throughout all the muscle-fibres small globules of fat were scattered.

The Fatty Heart and Anæsthesia.

A word may be said here with regard to "fatty heart" in connection with death under the administration of an anæsthetic. During a recent discussion upon death under anæsthetics one speaker, an authority no doubt upon one branch of medical science, but who, to the best of my belief, has not had wide pathological experience, rose and laid emphasis upon the statement that he had "again and again" found microscopical evidence of "fatty heart" on staining the heart-muscle with osmic acid. Such a statement would need qualification. If the instances referred to occurred in patients who were suffering from diphtheria at the time the anæsthetic was given fatty heart in all probability was found, but if the speaker intended to imply that the hearts of patients not suffering from such a disease had given evidence of "fatty heart," I feel it may be said that either preconceived ideas, or the fact that he used osmic acid instead of a far more satisfactory stain such as sudan iij, must have misled him. The view that "fatty heart" is a common cause for death under an anæs-

thetic I have no hesitation in saying is a myth. There are, it is needless to say, exceptions to every rule, and it is possible that from time to time cases may be met with in which the heart presents the conditions referred to in the next paragraph where death had occured under an anæsthetic. Such cases do not, however, correspond to the ordinary descriptions given of the "fatty heart".

The Fibroid Heart.

It may be repeated that fatty degeneration of the heart, when existing as the most important pathological condition present, occurs as a complication of an acute or subacute disease, either outside or within the heart. Fatty degeneration may, however, be associated with a chronic affection of the heart-wall. This affection is one in which sudden death may occur during apparently perfect health. At the autopsy, fatty changes, however, if present, are generally slight compared with the amount of fibrous tissue. *Fibroid disease of the heart, not fatty degeneration, has long been recognised as being very commonly found in cases of sudden death.* Some fatty changes are not infrequently also present, but the fat is only a stepping-stone towards the replacing of the muscle-fibres by fibrous tissue. Fibroid degeneration of the cardiac muscle is often consequent upon defective nutrition of the heart-wall due to interference with the circulation through the coronary arteries. It was pointed out, when speaking of sudden death in aortic valvular disease, that the most serious cases

are those in which there is disease of the first part of the aorta causing obstruction of the orifices of the coronary arteries. It was also mentioned that sudden death may occur when the aorta is diseased, while the cusps of the aortic valve remain healthy and competent. The association of disease of the first part of the aorta with fibroid disease of the cardiac muscle is perhaps the most common pathological condition found in cases of sudden death when death has overtaken an apparently healthy adult engaged in his daily occupation. This is especially the case when the adult is still in middle life.

In Fig. 10 the association of disease of the aorta with fibroid disease of the cardiac muscle is illustrated. The sketch represents a portion of the aorta and of the adjacent septum interventriculorum of the heart of a sailor, aged forty-six, who died suddenly. There was disease of the aorta which greatly obstructed the orifices of the coronary arteries and the cut surface of the muscular septum showed extensive fibrosis. The dark streaks seen in the sketch represent the muscle fibres ; the pale areas the fibrous tissue. Such well-marked fibrosis as this, is, however, exceptional.

In later life another cause of fibroid disease of the cardiac muscle becomes more frequent. Atheromatous disease may affect not the orifices of the coronary arteries, but the vessels themselves. Atheromatous disease of the coronary arteries is, however, common where the cardiac wall remains healthy, and occasionally there may be fibrosis of the muscle where

there is no obvious obstruction in the arteries. Possibly in the cases where disease of the coronary arteries is absent the nutrition of the heart suffers

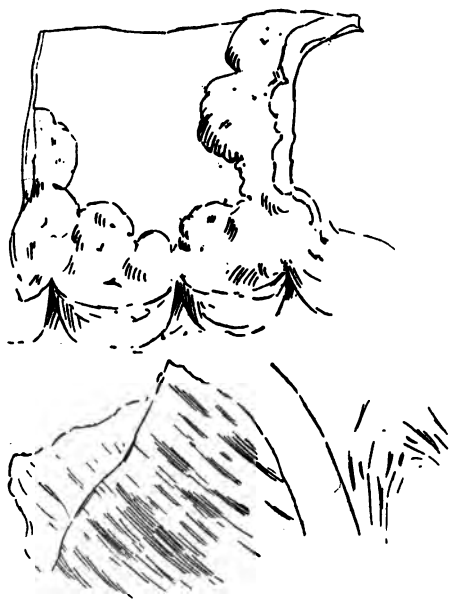

FIG. 10.—DISEASE OF THE AORTA OBSTRUCTING THE ORIFICES OF THE CORONARY ARTERIES.

Rough sketch of a portion of the heart of a sailor, aged forty-six, who died suddenly. Below the aortic valve a cut has been made, which exposes the interior of the inter-muscular septum of the ventricles, and shows that much of the muscle has been replaced by fibrous tissue.

as a consequence of old age rather than from definite local interference with nutrition. Another, but not common, cause of fibrosis of the cardiac muscle

should be mentioned; this is general thickening of the arteries throughout the body while, however, the nodular patches known as atheroma are absent. When this general thickening of arteries is present I have seen the cardiac muscle thickly studded with small circular patches of fibrosis resembling grey tubercles. Such an appearance in my experience is not common, but Professor Delépine exhibited a good example in the museum of specimens at the annual meeting of the British Medical Association when held in Manchester in 1902.

In cases of fibroid disease of the heart associated with disease of the aorta, or with atheroma of the coronary arteries, the amount of fibrosis of the cardiac muscle varies greatly. In some cases the fibrosis is so extensive that it is difficult to understand how the heart can have continued to act efficiently up to the time of sudden death; while in others the scattered fibroid patches are so small and so few in number that, allowing that they represent the extent of the disease, there seems nothing to explain the abrupt arrest of cardiac action. The fibroid patches may occur as small spots or narrow streaks numerous and widely distributed, or may exist as one or more large patches measuring one inch, or even nearly two inches, across.

Aneurism of the Heart.

There are other causes of fibroid disease of the heart besides disease of the first part of the aorta and atheroma of the coronary arteries. Syphilis may

give rise to this affection of the heart both through the medium of disease of the arteries and gummata. It has never fallen to my lot to meet with a case where fibrosis of the heart appeared to have followed disease of the coronary arteries of clearly syphilitic nature, but large fibrous patches which with little

FIG. 11.—A SMALL ANEURYSMAL BULGING AT THE APEX OF THE LEFT VENTRICLE.

doubt have been the sequel of the presence of a gumma are not very uncommon. I have seen a very large patch of this character in the heart of a prostitute, who, however, did not die suddenly. A fibroid patch is seen at the apex of the left ventricle, in Fig. 11, which may have been the sequel of a

gumma, but in this situation a fibroid patch is as often secondary to atheromatous disease of the coronary arteries. Fibrous destruction of the heart-wall, the sequel of a gumma, may occupy a very much larger area than this, and is almost as likely to be situated near the base as at the apex of one of the ventricles. The fibroid area generally bulges outwards, forming what is known as an aneurism of the heart, which in rare instances may rupture. Sudden death most frequently occurs, however, without any such rupture taking place. Yet not only these large patches, the sequel of gummata, but gummata themselves, in their earlier stages, may occasion sudden death.

A Case of Stokes-Adams' Disease.

It may be interesting to refer to a case of gummatous disease of the heart which came under my notice where death, although not absolutely sudden, was of acute onset. On calling one afternoon at a hospital in order to see one of the residents, I was taken to see a case which had recently been admitted. The patient, a finely built captain of a ship, was said to have had several fits, and one of these fits occurred soon after we entered the ward. He became suddenly unconscious and intensely cyanosed. No pulse could be felt, and no cardiac sounds were audible. There was a deep respiration about every half-minute, sometimes, perhaps, at a shorter, sometimes at a longer, interval. After four or five minutes had elapsed, during which time it looked as if every moment was

to be his last, he became flushed instead of cyanosed and the heart could be felt beating forcibly. The pulse was then about eighty to the minute and for a time regular. The impulse, however, soon grew feebler and the pulse irregular, the intermittence, however, varying with the respiration, occurred for a time at regular intervals, every fourth beat being absent. After another few minutes had passed the pulse grew regular, but was slow—only forty-four to forty-six to the minute. During this time the respirations had become natural, but the patient was restless and worked himself into the sitting posture. A little later, about twenty minutes after the onset of the fit, he was semi-conscious. On examination of the heart a loud widely conducted pulmonary systolic murmur was now audible, and also at the apex the dull diastolic sound, which sometimes has not inaptly been called "the third sound" of the heart. Several similar fits followed during the course of the evening and the man was dead before the morning. At the post-mortem examination a gumma was found in the septum interventriculorum immediately below the aortic valve. It was the size of a large filbert-nut and projected into the cavities of both ventricles. For many years it has been noticed that lesions of the septum interventriculorum are more likely to occasion serious disturbance of cardiac action and sudden death than areas of disease elsewhere in the cardiac muscle. Apparently, however, only comparatively recently has the possibility of interference with the bundle of His, which passes

from the right auricle to the septum between the ventricles in this situation, been thought to be the explanation of these disturbances. The above case is a good illustration of the sensitive character of the upper portion of the septum between the ventricles, and also of the combination of symptoms which are known by the name of Stokes-Adams' disease.

A lesion of the heart-wall was present here, which led to death, not suddenly, but in the course of a few hours. Death, however, may be absolutely sudden. A doctor whom I once met said he was talking to his father, who was preparing to start on his morning's professional round, when he fell dead, and that to all appearance he was dead before he reached the ground. In other cases there may be a short ejaculation indicating a sense of apprehension of evil, or death may be preceded by a sense of faintness. Again, a condition of collapse may exist somewhat resembling the result of an abdominal injury, or there may be distressing attacks of severe cardiac pain. Whether death be absolutely sudden or delayed for a few minutes, or even hours, some variety of fibroid disease of the cardiac muscle will, in the majority of instances, be found after death. There are, however, exceptions to this rule. Occasionally, especially in very old people, the heart will present no obvious lesions. In such cases cessation of cardiac action is probably due to the age of the heart-muscle. It has been unable to withstand some slight strain, or possibly disturbance by some toxin, which at an earlier period of life would have been harmless. But

even in middle life it may occasionally happen that the heart may fail, yet nothing abnormal can be detected. On one occasion I was asked to perform a post-mortem examination on a man, aged about thirty-five, who had returned home, saying that he did not feel well, sat down in a chair, and died. Nothing whatever could be found in the heart or elsewhere to account for death. Possibly such cases of cardiac failure are toxic in nature.

The Enlarged Heart Without Noteworthy Disease of the Muscle Wall.

Stress has been laid upon the fact that fibroid disease of the heart is the most common cause of sudden death. This is well recognised, but it is perhaps not so well known that in cases of sudden death nothing but a large heart may be found in which disease of the cardiac muscle and of the valves is absent. It is these cases which are most likely to be considered instances of death from mitral regurgitation. To those who are unaware that large hearts of this character may fail suddenly it is easy to commit the error—referred to previously—of mistaking normal thickening of the edge of the mitral valve for disease, especially as some text-books speak of mitral regurgitation as a cause of enlargement of the heart.

The Alcoholic Heart.

Occasionally the large heart may be found associated with a red granular kidney, but more commonly the kidneys are healthy. The most frequent

cause of a large heart, where valvular disease and chronic interstitial nephritis are absent, is alcoholic intemperance. Cases of sudden death are by no means uncommon where, although we may be ignorant of the history of the deceased, circumstances suggest that this is the most probable cause for the enlargement. For example, a carter, aged between thirty and forty, falls dead off the seat of his waggon while driving. A large heart is found weighing 16 ounces, but lesions of the valves, disease of the cardiac muscle and of the kidneys are absent. In such a case as this the occupation of the deceased man suggests that over-indulgence in alcohol was the probable cause of the enlargement. In other cases, when death is not absolutely sudden, the condition of the dying man may possibly incorrectly lead the police to think that he is intoxicated. As an illustration a case of a man, aged thirty, may be mentioned who was found "drunk" by the police. He died about half an hour later, and at the autopsy examination of his heart showed it to be much enlarged and weighing eighteen ounces; but no cause could be found for the enlargement. Here alcohol may have been the cause of the enlargement, but recent heavy drinking was not the reason of rapid failure. In connection with such a case, although the supposed drunken man proved to be dying of cardiac failure, it may be interesting to mention that a weak heart may fail while a man is taking alcohol. For example, a man, aged sixty-four, became collapsed while drinking in a public-house, and died very shortly after.

His heart proved to weigh as much as twenty ounces, but in this instance red granular kidneys were present, and the cardiac enlargement was probably not consequent upon alcoholic intemperance.

Hard Work as a Factor in Enlarged Heart.

To return to the cases of enlarged heart in which renal disease is absent and there is no valvular lesion or fibroid degeneration of the cardiac muscle, although in cases of this nature where sudden death occurs, there are rarely means of ascertaining the part played by alcohol in producing the enlargement of the heart, alcohol is without doubt the most common cause of such enlargement, and in any individual case what cannot be proved may be considered highly probable. There are, however, other causes for the enlargement, one of which is arduous work. Some instances which have come under my notice seem to indicate that it is not laborious exertion merely, but such exertion in a hot atmosphere that is likely to cause the development of these large hearts. At least the best-marked examples I have met with have occurred in two instances in gas-stokers, and in a third in a blacksmith. Fig. 12 is a photograph of the large heart of a gas-stoker, and a heart of normal size placed by its side for comparison. The heart of the gas-stoker weighed thirty-five and a half ounces. In none of these three cases, however, was death very sudden, but in one death took place in about thirty-six hours from the onset of the symptoms, extreme cyanosis being a marked feature. It may be

interesting to mention in this connection that the report of an inquest in a daily paper once attracted my attention, which mentioned that a striker in engineering works, aged thirty-four, had fallen dead while playing cricket, having just hit the ball for five runs. It occurred to me—although, since no post-mortem examination was made, it is needless

FIG. 12.—THE LARGE HEART OF A GAS-STOKER.

The smaller heart is of normal size, and is placed by the side of the larger for comparison. The age of the gas-stoker was forty-four.

to say there was no proof of the truth of this view of the case—that the man may have possessed a heart enlarged as the result of hard work in the hot atmosphere of the factory in which he was employed. Allowing that arduous work is most likely to deleteriously affect the heart of a labourer when he is under the influence of external heat, we may sup-

pose that the enlargement of the heart is in great part toxic in its origin. Nearly all the water drunk by the labourer passes off by the skin, and harmful products of metabolism are retained which should find their exit through the kidneys. These poison and weaken the heart which enlarges under the influence of physical strain.

A rare but remarkable cause of death is rupture of the heart. This cause of death seems to be especially common in insane patients. Mr. Cecil Beadles has recorded several such cases, which occurred in Colney Hatch Asylum. The cause of the rupture in some instances does not seem to be clear, though in others it is consequent upon local degeneration of the cardiac muscle due to interference with the circulation of a coronary artery.

Another uncommon cause of sudden death is thrombosis of a coronary artery. Curiously enough, as Dr. Parkes Weber points out in a case which came under his notice, and I believe the same observation had previously been made by Dr. Moxon, there may be evidence in the thrombus that life has continued sufficiently long after the thrombus has formed to allow the clot to become partially organised. I have, however, met with a case where both coronary arteries were thrombosed. It is needless to say that death must have followed the thrombosis quickly in this instance, though probably careful examination would have shown one artery to have been thrombosed before the other. Still another cause of sudden death may be mentioned, of which Dr. Osler and

others have recorded instances; this is, the detachment of a ball thrombus from the appendix of one of the auricles and its fixation in the corresponding mitral or tricuspid orifice. Although no such accident happened in the case from which Fig. 13 is taken, a

FIG. 13.—BALL-THROMBUS IN THE APPENDIX OF THE RIGHT AURICLE.

glance at the illustration will make it clear how easily the circulation could be stopped by the presence of the ball-like clot in the tricuspid orifice which lies below.

ANEURISM OF A SINUS OF VALSALVA.

ONE other cause of sudden death may be mentioned, because, although the disease which occasions it is not within the heart, it lies within the boundaries of the pericardium. This cause of death is aneurism of a sinus of Valsalva. Such an aneurism produces no symptoms until it bursts, and when it bursts into the pericardium it leads to very rapid death. Occasionally an aneurism in this situation may burst into the superior vena cava or pulmonary artery, and when this takes place death may not occur for some weeks, or perhaps a longer period, after the accident. In such cases interesting murmurs may be heard during life, but these do not concern us here. As an example of rapid death, the following case may be mentioned. A man, aged forty-six, awoke at 4 A.M. complaining of pain in the chest. He became collapsed and died at 7.30. At the autopsy an aneurism the size of a walnut was found in one of the sinuses of Valsalva which had burst in the pericardium, where 15 ounces of blood were present. An illustration has been given of one of these small aneurisms (see Fig. 14). In this case, curiously enough, although absolutely sudden death occurred, bursting of the

aneurism was not the cause. The possible mistakes of others have been mentioned, and I must confess my own. The specimen was removed from one of the first cases of sudden death upon which I made a post-mortem examination. Although aware of the

FIG. 14.—ANEURISM OF A SINUS OF VALSALVA.

The letter A points by a dotted line to the cavity of the aneurism. The specimen is from a man, a street hawker, aged thirty-eight, who died suddenly.

importance of fibroid disease of the heart, I did not then realise the frequency with which disease of the aorta, by causing interference with the circulation through the coronary arteries, leads to degeneration

of the cardiac muscle. The specimen was mounted without extensive cutting into the cardiac muscle. The heart has remained untouched since, but there can be no doubt that the cause of death is to be found in the muscle-wall. While, however, this aneurism did not rupture, it is a good illustration of yielding of the aorta over a small area immediately above the valve, and from its size it must be clearly realised how such a serious condition may exist without giving rise to symptoms, and the first indication of its existence be rupture ending in rapid death.

CONCLUDING REMARKS.

THE preceding observations have dealt almost entirely with sudden death as viewed from the post-mortem standpoint. They have been in great measure intended to aid those with little experience of morbid anatomy who may be called upon to investigate a case of sudden death. Medical men, however, may possibly in the course of a long career of practice meet with few such cases, yet they will be consulted by numerous patients who, at least for a time, live in daily fear that their days may be abruptly ended. To such patients, speaking generally, the teaching of the post-mortem room may be said to afford comfort rather than anxiety. Disease of the heart discovered during life comparatively rarely ends in sudden death.

In young patients, rheumatic disease of the aortic valve occasionally, but rarely, ends in sudden death, and such a termination to a case of disease of the mitral valve is so exceptional that its possibility is scarcely worth consideration.

When adult life is reached the most dangerous forms of disease of the heart are those which result from transgressing the laws of morality or of wisdom.

The disease of the aorta which leads to degeneration of the heart-wall by obstructing the orifices of the coronary arteries is with little doubt nearly always due to syphilis, and it is this disease of the aorta, either existing alone or in association with disease of the aortic valve, that is the most common cause of sudden death in early middle age. During the same and at a rather later period of life alcoholic intemperance manifests its effect upon the heart and not very uncommonly leads to abrupt cardiac failure. In this connection it may be mentioned that when making a post-mortem examination upon a case of sudden death no stress should be laid upon the absence of cirrhosis of the liver when the question of the probable part played by alcohol is being considered. Cirrhosis of the liver is the exception not the rule where there has been over-indulgence in alcohol.

In later middle age and in old age the heart not infrequently fails in consequence of disease for which the patient cannot be said to be in any way responsible. Atheroma of the coronary arteries, or merely senile changes in the cardiac muscle may lead to sudden death. Occasionally also the heart abruptly fails where red granular kidneys are present.

Although in the majority of cases of sudden death there will be one of the above-mentioned varieties of cardiac disease, occurring possibly near the time of life indicated, it should be stated that now and again a case of sudden cardiac failure may occur at almost

any age for which no cause can be discovered. In very rare instances it is possible that excessive strain may paralyse a healthy heart, but more commonly, it is probable that a case of sudden death in a young adult where nothing abnormal can be found in the heart, is the result of the poisoning of the heart by toxins which have entered the body by means of food.